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Art. III.—*The Cause of Bitter Pit.*

By ALFRED J. EWART, D.Sc., Ph.D.

(Professor of Botany and Plant Physiology in the Melbourne University, and Government Botanist of Victoria).

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In his fourth report upon this "disease," Mr. McAlpine in summing up refers the origin of Bitter Pit to two causes, both connected with the supply of water.

The first cause is considered to be due to irregularities of growth, the pulp growing more rapidly than the network of vessels, "so that meshes here and there are not formed and the loss of water at these spots cannot be fully met by a fresh inflow of sap. This causes the cells to collapse, the sap reaches an injurious concentration, causing the cells to die and turn brown."

The second cause is supposed to be the exact opposite of this, namely that an excess of water causes so great an increase in the pressure within the cells that they rupture and die, producing the characteristic appearance of Bitter Pit.

In regard to the first cause, since it is a morphological change readily capable of demonstration, one would expect to find some evidence of its existence brought forward. No such evidence is given in the whole of the voluminous reports. The statement is a supposition of possibility not based upon actual observation, and immediately contradicted by observed facts. Bitter Pit spots occur both on and near vascular bundles with healthy cells existing in the tissue further out supplied by the same bundles. Any interruption of the vascular supply would naturally affect all the tissue supplied by the bundles. In some cases a layer of bitter pit tissue may spread over a square centimetre or more extent, and crossing the course of a whole series of bundles. Nevertheless healthy tissue supplied by the same bundles may exist beyond the affected area, showing that the death of the affected cells cannot be due to an interruption of the vascular supply.

In regard to the second cause, excess of water causing an increased pressure bursting the pulp cells, it may be noted that the pressure within the pulp cells depends upon the osmotic concentration of the cell-sap within them, and that an abundant supply of water simply enables the full osmotic pressure to be exerted. In a

tissue, an increase of osmotic pressure simply causes the cells to press more firmly against one another and the tissue expands as a whole, sometimes leading in the case of thin-skinned apples floated on water to the bursting of the outer skin, but not to a bursting of the pulp cells. A bursting of the outer skin does not accompany the development of Bitter Pit, which on this theory it should do.

In addition apple pulp may be immersed in water without the cells being at first injured or bursting. Later they may die and brown, and in soft fleshed apples the pulp cells may separate singly or in clusters, but again without bursting, although here the supply of water is greater than ever exists when the fruit is on the tree. Both the bursting and the vascular interruption theories fail entirely to take account of the actual sequence of events in the development of Bitter Pit. In the early proteid stage of the apple, no signs of the disease are shown. During the starch stage, in certain localized areas the starch grains remain undissolved in groups of cells, which are at first colourless and living. These cells later die, turn brown and may collapse or shrivel. The first symptom of the disease is the non-solution of the starch grains. Now to prevent a dry starch grain from absorbing water a pressure of 2500 atmospheres is necessary, so that a pressure at least approaching this would be necessary to prevent it dissolving under the action of an enzyme, such as diastase, which can only act in the presence of water. The maximum pressure of the ascending sap is less than 1 atmosphere and the maximum osmotic pressure in apple cells containing 10-15 per cent. of sugar is 12 to 20 atmospheres. It is well known that in cells with quite as high osmotic pressures as this starch grains dissolve readily, and actual tests have shown that bitter pit tissue is more deficient in sugar than the ordinary pulp, which is the direct result of the non-solution of the starch grains. To invoke changes of sap pressure or of osmotic pressure as a cause of bitter pit is therefore a transparent absurdity.

Mr. McAlpine (p. 73) states "the theory of spraying with poisonous compounds was brought forward as a direct and definite cause. That theory has now been abandoned and the absorption of poison from the soil in infinitesimal quantities through the roots substituted for it." In justice to Dr. White who first put forward the poisoning theory of Bitter Pit, so misleading a statement cannot be allowed to pass uncontradicted. In the paper in question[1] Dr. White sums up in the following words: "The results of my

1 Proc. Roy. Soc. Victoria, 1911, p. 16.

observations seem to indicate that the complaint known as bitter pit is, strictly speaking, not a disease at all, but rather a symptom of slow local poisoning, and that in the cases actually examined so far it appears to be due to the poisonous compounds sprayed on to the surface of the fruits."

"Though as far as my observation and experiments go, I have limited them to the supposition that the spray passes through the breathing pores of the fruit, from the exterior, it is by no means impossible that some of the spray which falls on the ground or is washed down by the rain, and gradually accumulates in the soil, might enter the plant through the root hairs. That soil is capable of retaining arsenical compounds for a considerable length of time is well known, and when an orchard is sprayed 6 to 16 times in one season[2] the amount of poisonous spray reaching the soil must become quite appreciable. The sensitive fruit cells would probably be more susceptible to poison than the other parts of the plant, since the living protoplasm is reduced to a mere attenuated film, but this would necessarily be a matter for future investigation."

The statement that Dr. White's original theory has been abandoned and another substituted for it is therefore highly misleading. The essential point is that the complaint known as bitter pit is not a disease but a symptom of slow local poisoning, and evidence in support of this is steadily accumulating. The nature and origin of the poisons in question is a subsidiary matter. Dr. White informs me that as the result of several years' work on testing the influence of poisons on Prickly Cactus and other plants, she has come to the conclusion that bitter bit symptoms are not confined to such fruits as the apple and pear, but may also occur in leaves and stems, and as in the apple and pear may not only occur naturally but may be produced artificially by the direct application of poisons. It is indeed possible that certain obscure plant tumours and malformations may be the direct or indirect result of oligodynamic poisoning.

Potatoes grown in newly cleared acid soils often develop brown spongy patches of dead tissue internally which are not accompanied by any disease organisms. This and the disease known as "brown fleck" may be further instances of natural oligodynamic poisoning. The dead cells here also contain undissolved starch grains in quantity, whereas in dead patches produced by parasitic

2 Largely as the result of Dr. White's work spraying is no longer carried to the absurd extremest hat were formerly common, and it is more generally recognised that the smaller the amount of poisonous material used to produce the result required, the better it is for the plant and for the soil.

organisms, the starch grains are dissolved away. If metallic poisons are responsible for these defects, their presence should be capable of demonstration by direct analysis, since potatoes are much less sensitive to metallic poisons than are apples. Organic poisons are, however, also a possible cause, for it is now well known that the infertility to crops of certain peaty and other soils is due to the presence of traces of organic poisons of the nature of phenols, or creosote-like combinations.

The late Dr. Rothera carried out some experiments in the direction of endeavouring to produce bitter pit symptoms by the direct absorption of dilute solutions of metallic poisons. Cut leafy branches bearing fruit were placed in solutions of copper sulphate and mercuric chloride of strengths varying from 1 in 100,000 to 1 in 600,000. The results obtained were negative. The tests, however, only lasted a fortnight, and were discontinued on account of the shrivelling of the apples. The latter is due to the rapid blocking of the cut surface preventing the absorption of water (and poison). If the experiment is carried out differently by driving in the poisonous solution under a head of 5 or 6 feet of water or roughly one-sixth of an atmosphere positive results are easily obtained in a few days, and although the browning usually takes place along the veins, it is often strikingly reminiscent of bitter pit. When these tests were performed in the unripe starch stage, the apple always died before all the starch grains had dissolved in the unpoisoned pulp cells, but I have already shown that if dilute poisons are injected into young apples while on the tree, although most of the treated apples usually fall, some remain and ripen on the tree. These show bitter pit areas at the points injected and these areas show every sign of normal bitter pit, including the presence of starch grains in brown cells, while the surrounding healthy tissue contains no starch.

A second test carried out by Rothera was by watering apple trees with solutions of copper sulphate of 1 in 100,000 strength.

The following results were obtained :—

	Yield.	Pitted.	% Pitted.
Counted by Mr. McAlpine—			
Gravenstein -	100 -	8 -	8
Jonathan - -	164 -	16 -	9.7
Counted by Dr. Rothera—			
Charles Ross -	48 -	10 -	20.8

Mr. McAlpine, however, states that the trees used were previously liable to Bitter Pit. Why such trees were selected for the test is difficult to say. To be conclusive the tests would need to be repeated

on a larger scale over at least 2 years, and on trees whose previous history was known.

The present position in regard to the theories of the origin of bitter pit may therefore be summed up as follows :—

(1) The bursting cell theory is founded on a confusion between cause and effect. The cracks in the cell walls sometimes seen in sections of dead bitter pit tissue are either cracks caused after death by drying and contraction, or more usually are tears produced by the razor. In sections cut from tissue imbedded in paraffin the cracks are rarely seen, and are often entirely absent.

(2) The vascular interruption theory is unsupported by any morphological evidence, and is contradicted by direct evidence such as the existence of healthy tissue beyond bitter pit areas where according to the theory vascular interruption should have occurred. Further, the occurrence of starch in the cells shows that the channels along which carbohydrates are conveyed to the bitter pit tissue must have been open and functioning freely.

(3) The poisoning theory has behind it the weight of the following evidence :—

Every symptom of this defect can be produced by the artificial application of poisons, including the presence of starch grains in dead cells.

Various observers have noted similar results in leaves and young stems as the result of the application of poisonous sprays (patches of dead tissue with brown shrivelled cells packed with starch.) In apples the sensitivity is so great that the poisoning may be oligodynamic, i.e., poisoning may occur in the presence of traces of poison beyond detection by ordinary chemical analysis.

As apples ripen the sensitivity of the pulp cells to poison increases, so that an apparently sound apple may develop bitter pit after it has been picked.

There is a close correspondence between resistance to poison and resistance to bitter pit. The most resistant variety to bitter pit (Yates) is also the most resistant to poison. Varieties specially sensitive to poison are also specially sensitive to bitter pit. There is also a close correspondence in regard to temperature effects. At low temperatures the development of bitter pit is checked or retarded. Similarly at low temperatures the resistance to poisons is greatly increased, being 10 to 100 times greater at 0°C what it is at 25°C.

Further, there is evidence to show that where a comparison is possible the incidence of bitter pit is greater in orchards that have

been heavily sprayed for some time than in orchards which have been lightly sprayed or not sprayed at all. It is unfortunate that statistics on this point, which by now should have been available in abundance have not been published. It is quite beside the point to indicate the existence of bitter pit in unsprayed orchards as a complete and satisfactory answer. If bitter pit is simply the result of local oligodynamic poisoning, any trace of any poison capable of absorption may produce it, independently of how it is absorbed and whether it is originally present in the soil or not.

The Browning of Bitter Pit Tissue.—In certain varieties of apples, notably Statesman, the pit tissue is usually paler than in other varieties. The browning is due to the action of the oxidase liberated from the dying protoplasm upon the tannic acid of the cell sap. When a Statesman apple is cut, the cut surface remains paler than in most apples, the browning being most evident along the veins. This might be due either to a deficiency of oxidase or of tannin or to the presence of an oxidase inhibiter. More than one distinct variety of apple has been known as Statesman. One of these false Statesmans is known as Chandler's Seedling. Its pulp browns readily when cut. The sap was expressed from equally ripe true Statesman and from Chandler's Seedling and tested for tannic acid; the former yielded 0.24 and the latter 0.16, a difference which is hardly sufficient to explain the pronounced difference of browning.

Testing with guiacum and peroxide of hydrogen for oxidase gave about the same depth of blue, but this test though a delicate one does not discriminate quantitively. A dilute solution of amidol is a good test for apple oxidase. By adding varying quantities of apple sap until the rate of change of colour was the same in each case, it was found that three parts of Chandler sap contained as much oxidase as 8 parts of Statesman sap. The glycerine extract from pulp dried by pressure gave values of 3 and 9 respectively. No evidence of the presence of any special inhibiting agent could be obtained, so that the feeble browning of Statesman pulp is mainly due to a deficiency of oxidase. The amount of oxidase present in an apple can therefore to some extent be used as a test to distinguish between certain varieties.